175

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## Editorial

In this issue of The Veterinary Journal are published a number of articles of topical interest and considerable importance.

The subject of *Specific Ophthalmia* is dealt with by Lt.-Col. H. Greenfield. As Greenfield points out, this disease in the horse is mostly found under war conditions and only when such conditions prevail for a considerable length of time. Many possible causes have been described from time to time in different countries, but little of a definite nature has been found to explain the changes in the eye associated with the typical affection. Those of us who had experience of the condition during the Great War will recognise many of the associations referred to in the article and will agree that

XUM

the conclusions arrived at by the author coincide to a considerable degree with our experiences. The occurrence of the true disease in the camel is of much interest and adds largely to a clearer understanding of its pathology and etiology. These observations come at a very appropriate time, for it may be that the horse will be more largely used both at home and abroad under conditions which might tend to encourage the development of specific ophthalmia. If the adoption of suitable prophylactic measures as suggested by a perusal of the article is followed by a lowering of the incidence of the affection, a step forward will have been made in the understanding of its causation.

From Dr. Russell Greig is an important contribution on *Scrapie* in sheep. This disease has been the subject of research for some years in Scotland and in certain parts of Europe. From the data now available there seems to be definite evidence that Scrapie is an infectious disease and that the infection can be conveyed through the medium of the pasture. Evidence is also produced elsewhere that the disease can be reproduced artificially in susceptible sheep. The advance in our knowledge of the maladies affecting sheep has been rapid and the patient research into Scrapie has resulted in an extension of our understanding of a disease which for so long seemed to yield little information of a definite character. These results are one more example to be added to the list of achievements of research by members of the veterinary profession.

The case of *vitamin C deficiency* in the young dog is of more than passing interest. The association of the various vitamins with the health and disease of the domestic animals is gradually becoming better understood. The feeding of animals was never more important than at the present time, in view of the substitutes that may have to be used and the early recognition of the symptoms accompanying any deficiency of essential elements—vitamins or other substances—is essential in order that the necessary correction may be made in time. It behoves the veterinary practitioner to become acquainted with the latest observations on this vast subject in order that he may be in the position not only to repair damage that may be done but to act in an advisory capacity to agriculturalists and others on food and the nutritive value of substitutes which may have to be used.

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## PERIODIC OPHTHALMIA

THE disease of the horse's eye commonly called Periodic Ophthalmia has always been a subject of great interest to veterinary surgeons. It would appear to have been a relatively common condition during the last century and also one whose incidence was greatly increased when horses were collected

together in large numbers and especially so if such collection took place under what might be called unfavourable circumstances.

In pre-mechanical days it was a scourge in the great industrial stables and also in army animals. Its incidence increased if horses were kept under bad hygienic conditions and also when they were subjected to the hardships of a campaign.

Much has been written from the clinical point of view and the morbid anatomy of the lesions has long been a more or less open book. As yet, however, its true etiology is unknown and its treatment has been non-specific and based on the hit and miss principles of the alleviation of symptoms. The very nature of the disease inclines one to the belief that it has some specific origin and yet in spite of the efforts of investigators of repute in many countries its cause remains a mystery. The disease has certainly reduced in its incidence during the last 30-40 years if one accepts the reports of old practitioners. There are some who say that the cases which are reported nowadays are not true periodic ophthalmia. Be this as it may, there is no doubt that during the South African War, 1899-1902, and Great War of 1914-18 many cases of ophthalmia occurred on active service which resembled in every way the periodic ophthalmia which has been described by our forbears. Fortunately in recent years the disease has been almost conspicuous by its absence amongst the civilian animals. During the last few years, however, some observers have reported cases amongst imported animals. This has focused attention on the disease and has given rise to considerable discussion.

Judged by previous experience, war time is a favourable period for the visitations of this disease and for its spread in epidemic form. It leads to great loss in efficiency and on the economic front.

It is hoped that the article by Lt.-Col. H. Greenfield which we publish this month will reawaken an interest in this intriguing disease.

The essayist speaks with the authority of one who has extensive personal experience of the subject which he debates and has made a critical study of the bibliographical records.

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XUM

## General Articles

# IRIDO-CYCLITIS (Ophthalmitis) AMONGST ANIMALS AT WAR

By LT.-COL. HERBERT GREENFIELD, O.B.E., M.R.C.V.S.
*Camberley.*

### Synonyms

PANOPHTHALMITIS infectosa (toxica) recidiva equi, recurrent iritis, irido-cyclitis, irido choroiditis, irido-choroiditis recurrens, periodic, recurrent, remittent, rheumatoid, specific or constitutional ophthalmia or more modern ophthalmitis, uveitis, moonblindness, lunatic disease.

### Definition

The disease is an inflammatory condition mainly affecting the inner structures of the eye, especially the uvea, which shows a marked tendency to recur and to be progressive until sight is reduced or lost owing to destructive changes.

### Nature

Some of the oldest observers (Wardrop, 1819, Sichel, 1862) recognised that the primary lesions existed in the iris, but certain clinical characteristics have been allowed to effect the nomenclature, tending to hinder the differential diagnosis and recognition of the disease. None have done this more than the generalisation due to its being loosely diagnosed ophthalmia, and regarding it as a disease peculiar to the horse, and as a specific one.

The term ophthalmia was probably coined to cover inflammation of the eye in a remote past. There is now no ophthalmia as such recognised by ophthalmologists; certainly no disease including inflammation of the deeper structures of the eye in the human subject with a similarity to that among animals. It is not a condition peculiar to horses. A similar disease is common to man and cattle. It has been seen in dogs and birds. The writer has observed large numbers of camels affected in Waziristan identical in every respect to "specific ophthalmia" in horses. Iritis invariably tends to be recurrent.

Greenfield[14] showed that the serious part of ophthalmia in the South African War (1901-1903) was an iritis.

Textbooks, including those by Nicolas,[17] Wooldridge,[8] Jakob,[20] Official History of the Great War,[19] appear to accept fully that the various forms of ophthalmia are due to an inflammation of the uveal tract, mainly affecting the iris; so that it might be described as uveitis, irido-cyclitis, or, in short, iritis.

It may be inferred also that the condition is regarded as representing the most important of serious eye diseases.

Iritis in domestic animals is generally sporadic in its incidence, frequently enzootic and but rarely epizootic.

The outstanding feature of its occurrence in modern times is its scourge-like proportions during prolonged wars.

So significant is this and some facts relating to it, that it is proposed to treat the disease from this aspect. For it appears justifiable that if the views held concerning this disease do not stand the test of review in accordance with the experience of the large numbers who worked on it during the Great War, then these views should be revised. It was admitted by most, if not all, that the disease was specific ophthalmia, moonblindness, etc., as of older times. The statistics for the British Army during the campaigns to be considered are as follows:

| *War* | *Date of commencement* | *Date of 1st case* | *Height of incidence of iritis* | *Number and proportion of cases* |
|---|---|---|---|---|
| South Africa | Oct., 1899 ..... | Dec., 1901, 2nd year ... | 1903, 3rd & 4th years | Nearly 2,000, 14 per cent. (1). |
| Great War ... | Aug., 1914 ..... | Dec., 1916, 2nd year ... | 1917-18, 3rd & 4th years | 6,000 cases under treatment in hospitals (2). Some divisions in the field, 10 per cent. (3). |
| Waziristan ... | Animals joined Forces at various times | 1919-20, ? ... | 1920-21, 3rd & 4th years | About 1,000 camels affected, 10 per cent. (3). |

Authorities for the above are: (1) Nicolas,[17] (2) Major-General Sir J. Moore's report, (3) author.

### Development

Major-General Sir F. Smith, in his Veterinary History of the South African War,[13] first brought notice to the extraordinary delay in incidence, which he found to be inexplicable. The dates of development, both in the Great War and in Waziristan, are so identical that I submit for the first time that the only conclusion is that the development is slow or that it is dependent upon a train of circumstances which only accumulate sufficiently after a long period.

Some support to this conclusion is to be found in the only facts of incidence available on the point in peace time. Nicolas[17] says that a regiment of Hussars, among the horses of which 130 cases of ophthalmia had occurred at Hofgeismar, was sent to Frankfurt, and was replaced in 1875 by a regiment of Dragoons in which no cases of the diseases had ever occurred. Cases then developed as follows: 1876, 5 cases; 1877, 12 cases; 1878, 11 cases; 1879, 14 cases; 1880, 42 cases. Zundel, quoted by Law,[9] says that of 700 army horses stationed at Saarburg in 1878, six were attacked with recurrent ophthalmia in 1879 and 42 in 1880.

## Etiology

During peace, as in war, ophthalmia has occurred in larger or smaller numbers, under peculiarly dominating conditions. So marked have some of these been in their relationship to incidence, such as endemic conditions, damp climate, soil or stable, and heredity during peace, and others such as exposure, dust and glare during war, that they have unhesitatingly been proclaimed causes.

These will be dealt with now and be described as predisposing or contributory causes. It is believed that their role is generally a subsidiary one, partly because they are so contradictory and partly in that they do not appear sufficient in themselves to account for the subsequent pathology, and finally, because they do not agree with the experience of occurrence in war.

### Contributory Causes

1. *Endemic conditions.*—Throughout the history of both human and veterinary pathology there has been a continued readiness to associate the origin of iritis with rheumatism.

Doubtless that may be due in some degree to recurrence being confused with the origin of the primary disease (to be described later). Nevertheless, it is beyond doubt that ophthalmitis had existed in the main in damp, cold latitudes, and particularly cold lowlands, and this association has been responsible for its distinctive enzootic character in peace time.

Some observers have localised the disease and magnified the enzootic character of ophthalmitis of the past even more, and maintained that it belonged to the lowlands of North Europe, from which it was distributed to England and possibly other countries by troops engaged in the Napoleonic and other wars.

This is supported in some measure by the fact that large numbers of horses affected during the Great War were animals in this particular area or distributed from it.[19] On the other hand, it did not occur in horses distributed to warmer climates, e.g., Salonika, Egypt, or to England after the war.

This finding does not complete or cover the whole of the modern history of ophthalmitis. When it ravaged the army in South Africa it was spread

over a great part of that sub-continent, most of which is notoriously dry and healthy, and occurred amongst animals from many sources which had never known a cold, damp climate or lowland. Waziristan afforded even a more striking contrast, in that it was typically high and dry, and camels there could have experienced nothing else material to the point.

Not only have damp weather climates and soils been held culpable, but fodders grown in such places have been condemned by some authorities. This, however, will not stand the test of war experience, when most animals were fed on imported food, many of the affected entirely so, for the whole of the campaign.

2. *Heredity.*—Heredity occupied a position as strong in the etiology of this disease as in many others, to be similarly discarded with the increase of knowledge. The great widespread outbreaks in war amongst animals of all types, classes and origins must have eliminated heredity from any direct influence.

3. *Hygiene and Sanitation.*—The disease has been, and still is, believed by some to arise from basement and damp stables.

The Official History of the Great War[19] says that large numbers of stables in North America, even those used for high-class horses, had no light or sanitation, and that remounts from this district provided many cases, and that there it was attributed to lack of sanitation.

On the other hand, observers say that when it was prevalent in England it occurred as frequently in the best stables as those less favoured. Some maintain that coalpit ponies were never affected.

Officers' chargers and horses of small units, crammed into buildings, suitable or unsuitable, sanitary or insanitary, for shelter during the Great War, rarely if ever became affected, while it ravaged those around in the open. This is the definite conclusion of the author after continued observation on this point for at least twelve months.

Moreover, the camels affected in Waziristan, the mass of animals diseased in South Africa, and probably also those of the Great War (at the time they became affected) had never seen a stable or had not been housed for years.

These observations show that there is little or nothing to suggest that lack of sanitation or the congregation of animals "breeds" the disease.

### Seasonal Prevalence

Diversity of opinion exists as to the season of special prevalence. Bayer[5] considers it to be the summer, but generally, the records show that it is noticed mostly in the first and second quarters of the year, particularly the periods of changeable and equinoctial weather.

The point is not of definite importance until the period of development is known, which is highly problematical and certainly not decisive, if recurrence of the disease is confused with onset, as often appears to be the case.

XUM

### Cold and Exposure

In each of the three wars the affected animals were generally those longest in the country and those belonging to the largest units, and therefore those for which it was impossible to find cover or protection; moreover, it was most marked in the coldest and most exposed part of the country or countries occupied.

In the Great War, with all its many campaigns, it was reported in no hot country; it was confined to Flanders, France and North Italy.[19]

In Waziristan there were no cases in the hot plains of Dera Ismael Khan and Tank; the disease was to be found amongst camel corps with the Wana column and others working in high areas which were bleak and bitter in winter.

These weather conditions are to be found to the full in North China where the indigenous Mongolian ponies and mules live a similar existence to that of active service and suffer severely from iritis, especially the mules.

Care should be taken to eliminate the effect of cold and catarrh upon recurrence in estimating the effect of the weather upon etiology; a point to be discussed later. It should not be overlooked, however, that important, varied and considerable as are eye diseases in hot countries like India, Egypt, Mesopotamia and Palestine, little recognised iritis or ophthalmia is found amongst animals in peace or war.

The literature suggests a higher percentage of cases amongst army horses in peace as well as at war. This may be due to concentration and its risks; but the fact that coach horses, leading a life subject to similar exposure and change of environment, suffered considerably, while colliery ponies did not, supports the view of exposure rather than congregation being the cause.

The areas in which the disease existed enzootically for so long are the few exceptions, all being in cool or cold latitudes, and nearly all exposed to some rigours in winter. (See endemic conditions.)

### War Conditions

Various conflicting causes have been added to the foregoing as contributory conditions during war, amongst others, glare, flies and mud. They do not appear to have been sufficiently consistent to be very material. Eye fringes may or may not have modified symptoms by reducing glare in Waziristan, but glare can have had little effect in producing iritis during the gloomy winters in Flanders.

Although Major-General Smith proved that flies had a serious role in the production of keratitis in India,[17] he and others proved that their influence upon ophthalmia was negligible and that ophthalmia was not reduced by the use of eye fringes in South Africa.

Mud and wet conditions cannot be said to be common or dominant in the

three campaigns concerned, serious as it was in France. Ophthalmia is said to have disappeared in South Africa after the introduction of floored stables,[19] when the mud was eliminated, but by this time the animals were sheltered and living under peace-time conditions (conditions under which the disease had been unknown previously in South Africa). Moreover, a great part of the iritis in South Africa occurred on the high, dry veldt, while in the third campaign, Waziristan, it occurred in one of the driest districts in the world.

Having discussed the circumstances under which ophthalmitis occurs and finding that of the many reputed contributory causes, only one, cold and exposure, shows any effect in war and appears therefore noteworthy, we pass to agencies which can, do and will produce iritis.

**Definite Causes**

1. *Traumatism.*—Various forms of traumatism may induce inflammation of the uvea, including contusions and wounds, particularly wounds with infection, which will be discussed under exogenous infection.

2. *Diosmosis.*—Hyperæmia and inflammation of the iris may be set up by diosmosis, from irritating substances and chemically active foreign bodies or aggregations of bacteria, in the anterior layers of the cornea or conjunctival sac.

The number of cases produced directly in this way in animals may be small as compared with the large incidence seen at times, but the fact remains that iritis can be so produced and suggests that this process might have a significant share as a contributory cause in support of, or together with, others.

3. *Local extension.*—Iritis does occur definitely, though occasionally, from other local and neighbouring diseases of the eye in all animals, particularly those with continued or extensive infection. Usually the preceding affections are those of the anterior parts, but it may follow at times upon choroiditis, and at others, though more rarely, upon retinitis.

Many writers have observed that specific ophthalmia has been frequently observed to follow symptoms of minor diseases of the eye. Greenfield[14] went further and laid emphasis upon the fact that the history of many cases of ophthalmia showed that the earlier affection had been treated, if at all, in the open without shelter or shade to the eye, and considered that this was an important factor in the etiology of iritis in war.

This finds support from the remarks of Nicolas,[17] who, speaking of the time when ophthalmitis was grossly prevalent, says there was then a tendency in the French Army to regard eye affections as too trivial to warrant hospital treatment, and that up to 1902 an average of 130 horses were cast annually as blind, while no eye cases were admitted to hospital for treatment.

Doubtless this was an attitude by no manner of means confined to France. Moreover, older writers emphasise the occurrence of panophthalmitis (often

XUM

septic purulence), which also points to local, rather than a specific, infection.

Herein, it is suggested, lies a factor which is important in the two vital questions on the etiology of iritis to which no effective answer had been made, viz.:

Why is iritis so rare now as compared with formerly?

Why does it occur as such a scourge during war?

Iritis is largely the result of extension from other injuries and local disease, accentuated by exposure, lack of treatment and maltreatment (including treatment by irritants).

4. *Sympathetic iritis.*—In the human subject one of the most disastrous forms of iritis is a sympathetic condition, in which iritis of one eye occurs as a consequence of irido-cyclitis in the other.

In the first or exciting eye there is a plastic irido-cyclitis often associated with detachment of the retina, similar to that commonly found in the horse.

There is an irritation, photophobia, lachrymation and pain in the other eye at the same time.

This may be followed at once, or more usually after a period of 30-90 days or longer, by a secondary iritis in the sympathising eye.

In the latter, hypopyon is rare, but the iritis is recurrent, severe, and progressive and the sympathising eye is generally lost.

The affection of the exciting eye is always an irido-cyclitis (inflammation of both iris and ciliary body). It does not follow ulceration of the cornea, staphyloma or paraophthalmitis as a rule, if at all.

The method of transmission is unknown, whether it is through the optic or ciliary nerves or their lymphatics or by the circulation, or whether it is a form of anaphylaxis. It is considered by some to be analogous in many respects to protozoan diseases and the condition reacts somewhat to Salvarsan.[7]

The important points here are that the disease definitely arises from an external infected iritis, that it occurs as recurrent progressive iritis with many features in common with specific ophthalmia of horses and that it is not a specific disease.

Wardrop,[2] one of the best observers, says definitely that sympathetic iritis does occur in the horse. Greenfield[14] was of the opinion that the symptoms of ophthalmia in South Africa suggested it.

The author is convinced that ophthalmia as seen in war recurs in the alternate eye; Bayer, Percivall and many others noticed this in peace time.

5. *Metastasis.*—Amongst domestic animals this process occurs in such conditions as influenza of horses, malignant catarrh of cattle and distemper in dogs, but a significant point is that it is relatively rare. This is important as it has a bearing upon endogenous infection, for the fact that metastatic iritis is so rare in such diseases as strangles and distemper, whose distribution and metastasis are by no manner of means so restricted elsewhere, appears a

material and weighty argument against iritis arising from a systemic infection (endogenous).

6. *Focal infection.*—Modern research in human ophthalmology has advanced the importance of the origin of iritis from a focus of infection elsewhere. Search is now commonly made for pyogenic centres, e.g., by X-ray of teeth, etc. Commonly, the dormant pyogenic centre is in the vicinity of the eye, but this is by no means essential.

This is of interest to us, because the symptoms of this type of iritis in man are those most typical of the disease in the horse, while focal centres of infection, such as thrush, grease and cellulitis, are common amongst animals, especially in the conditions in which iritis prevails.

The larger incidence of iritis amongst camels in Waziristan was peculiarly interesting because the disease assumed a proportion of 10 per cent. among 10,000 camels, of which at least 300 went blind, while equal numbers of horses, mules and bullocks showed no evidence of the disease. The camels, which have good natural eye protection, suffered for the first time in such numbers, while locally employed civil animals appeared to be unaffected, and remained unaffected while the conditions of their work approximated that of their normal habit, country and condition (except the one to be considered) more nearly than did that of the other army animals.

The disease occurred amongst corps in which surra had existed and which were exposed to the rigours of the Indian Frontier winter. It did not occur among the corps formed entirely of animals segregated for surra in the hot plains of Dera Ismael Khan.

Unless this combination of trypanosome infection together with exposure and cold was sufficient to stimulate the origin of ophthalmitis (which condition had previously been commonly associated on the North-West Frontier of India without the recognition of similar ophthalmitis), it occurred with no known cause of iritis which the author could find or investigate, except focal infection.

The corps which furnished the affected animals worked daily through the rocky water courses from camp to camp, where their constant traffic through water and over rough river beds had softened and worn the footpads until they were subject to a condition akin to advanced thrush, units often having 20 to 25 per cent. of their camels unfit for work on this account.

This condition had been rife for probably two years before iritis occurred.

It is suggested here that this provides a definite clue to a link in the origin of specific ophthalmia. The disease occurred in the past when pyogenic diseases were little understood and pus was oft-times considered "laudable." War times involve horses in excessive risks of pyogenic infection (especially in cold, damp latitudes in which it occurs commonly) and from thrush, grease, wounds, etc.

The camels in Waziristan were affected with an extensive amount of iritis

for the first time on a Frontier subjected to many campaigns with well-recorded histories—the special features of this one being its duration and the foot disease.

In France and Flanders the focal infection area may have been well represented by the previous year's widespread outbreaks of ulcerative cellulitis and other skin diseases of the limbs due to mud.

This appears to bring the views of the etiology of equine iritis into agreement with modern research and seems to the author to furnish a valuable clue to the special prevalence of iritis in the past and at war.

7. *General diseases.*—The role of general and kidney diseases in the incidence of iritis in man has depreciated of late. In veterinary ophthalmology it has quite rightly occupied a position of little importance. Auto-intoxication can likewise be considered a possible but unlikely source.

Endocrine diseases may have an effect upon the iris. They may be relevant to our inquiry as being predisposing conditions, for ophthalmitis is prevalent only after prolonged wars and then at a season when endocrine diseases are equally assertive.

8. *Parasitic infection.*—In the past, parasitic infection as a possible cause has held a conspicuous place. Ophthalmia has been attributed to filaria, cysticerus, distomata, cercaria, malaria, trypanosomes, and many other parasites.

In countries like Burma, S. China and India, where filaria abound, iritis is virtually unknown amongst animals. In the widespread experience of the author many forms of ocular disease may be associated with them, but never iritis.

This also applies to F. oculi equi and F. papillosa which invade the aqueous humour and are now known to be practically harmless.

The class of animals which suffer from cysticercus and distomata generally suffer little, if at all, from iritis. While the camel of N. Waziristan, which suffered from trypanosomiasis, sometimes developed ophthalmitis, those segregated in a special Surra Corps at Dera Ismael Khan were noticeably free.

During the Great War iritis abounded in Belgium, France and Northern Italy, while it was non-existent in campaigns in many countries where piroplasmosis, trypanosomiasis, filariasis, etc., were rife in man and beast. Surely this should be sufficient to direct attention to other sources for the causation.

9. *Microbial infection.*—The outstanding contrast in veterinary and human ophthalmology is the continued search for an originating specific organism as the cause of iritis in the one, and the equally assiduous search for additional causative agents in the other.

Yet in iritis or ophthalmitis in the horse are signs and symptoms of ordinary inflammation as in other animals and in other sites. No contagion

can be traced or transmitted by any known means. The bacterial flora of the conjunctival sac in iritis show nothing abnormal.

The inference that it is not a purely specific disease is supported clinically during peace, as at that time it occurs sporadically and often in animals isolated and grazing, while similar conditions can be produced by the injection into the eye of many different organisms, even those of usually harmless saprophytic type such as B. subtilis.

At war it occurs largely in the animals most exposed in the open and probably only in those exposed for a long period. It has occurred little, if at all, during short campaigns, although conditions suitable for the development of specific disease may have been ideal.

It is, however, difficult to admit that such a large measure of inflammatory condition, localised though it may be in iritis, is unassociated with infection—this difficulty is accentuated when large numbers of cases occur enzootically or almost as an epizootic as during war.

Generally, the infection is regarded as endogenous; with this, however, the author does not agree. Veterinary literature shows that the iris of domestic animals does not suffer much from metastatic infection transmitted by the blood during specific disease, and that when such a type of disease is induced it is generally localised, slight, and does not often lead to atrophy of the eye. These are not the characteristic features of iritis in animals. Moreover, hypopyon is an outstanding symptom of equine iritis and contains a large proportion of polynuclear lymphocytes, suggesting exogenous infection.

It is submitted here that the dominant causative agent is an exogenous infection, accentuated by cold and exposure and by the effect of focal suppuration. The focal suppuration effects may be localised or attracted to the eye by the inflammation induced by the local infection or vice versa; the local infection being assisted by the lowering of the resistance of the eye tissue due to focal infection, toxins, etc.

Keber[20] has advanced the exogenous theory of infection by the thesis that, as saprophytic germs produce panophthalmitis when introduced by intraocular injection, and their admission through the frequently observed corneal injuries and more frequently unobserved ones is simple, so is the disease produced.

This appears a strong case for exogenous infection, but is not entirely conclusive. It does not account for the vast difference of incidence amongst animals at peace and at war.

The missing link is suggested as follows—normally the aqueous, which becomes involved from an extension from the frequent inflammation and infection of the superficial eye, is as indifferent a media for bacterial growth as the vitreous, which is rarely involved, is good.

This is due to two factors: the former is liquid and its flow through a

penetrating wound washes out the infection, and it contains a rather lower percentage of albumen.

The effect of the cleansing flow is lost when the traumatic injury is too small to permit of the egress of the aqueous, and it must be remembered in this connection that the small wound is generally more dangerous than the larger one. (Large wounds, and ulcerated cornea and staphyloma, may not be followed by serious infection.)[7] The percentage of albumen, etc., may be increased by environment, contributory causes and local diseases.

The cornea is usually an all-sufficient protection against exogenous infection, but during continued exposure or repeated superficial disease it may become infiltrated and liable to invasion. The sympathetic hyperæmia then causes an increase of the solids of the aqueous, and so the chain of ingress may be completed.

This chain of ingress is rendered more liable to use by war conditions. Under such conditions animals working upon partially made or unmade roads, as well as standing exposed to wind and weather day and night, are subjected to excessive action of dust and grit; they are often tied up facing the wind with no possibility of the eyes getting any protection. At times, the animals receive little attention as far as cleansing or sponging of the eyes is concerned (as little as probably received in the old times, when ophthalmitis was prevalent and widespread), and often in the cold weather the eyelids are reduced to a state of semi-paralysis, which is followed by a stasis in the drainage of the lachrymal fluid with consequent dacyrocystitis.

This theory of a slow cumulative chain of exogenous infection appears to agree with the clinical evidence that iritis in the horse follows a previous attack or attacks of superficial disease. (Schwarzenecker,[5] Greenfield,[14] Guard[16].)

It agrees with the view of slow development. It may also explain the varied course and relapses of the condition.

Numerous theories have been advanced to explain that the remission of symptoms in the course of iritis is due to chemical changes such as the rise and fall of reaction (acidity) in the vitreus, inhibiting the growth of a causal organism. It appears to be more likely that it is due to the changes consequent upon exudation (as advanced above), as is usual in all inflammation.

The influence of cold and exposure raises two points of possible interest which have received little, if any, attention, viz., that of infection or toxic absorption from the lachrymal sac and the effect of moulds.

### Lachrymal Sac Infection

At Lebouvriere, in May and June, 1918, during the Great War, the author had for a few weeks some 70-80 horses suffering from irido-cyclitis in semi-acute form but working with their units, under the experimental treatment of

syringing out the lachrymal passages to remove the possibility of re-infection of the eye from disease in the lachrymal sac.

An interesting point which may be of value was that amongst these animals the lachrymal passage was blocked in 20 to 30 per cent. of cases. In approximately 15 per cent. the occlusion had been of such duration and suppuration was so intense in the blocked channel that there was ulceration through the lachrymal duct beneath the eyes or, more commonly, through the wall of the nasal duct and into the nose. In at least two cases there was ulceration through the facial bones and on to the face, incredible as it may appear. (These facts and figures have been supported by similar observations in other cases since that time.)

The lachrymal ducts were syringed out every other day and exact records kept of the lesions, progress, etc., but it is regretted that it is only possible to give approximate figures from memory. Enemy action enforced the termination of the treatment, and the records were lost during later operations.

As there is no considerable discharge or condition during iritis which would produce such extensive inflammation of the lachrymal passages, it seems possible that the lachrymal disturbance may precede or actually cause the iritis. We know from continued observation that nasal catarrh will produce a recurrence of iritis; this finding is somewhat confirmatory.

### Moulds

Investigation as to whether moulds might play a relatively important role in the incidence of iritis may be worthy of attention. The disease has been attributed to endemic conditions associated with lowlands, damp climate, damaged grain, food grown in marshes, etc., and such an origin would not be opposed to the history of the disease in war, as it occurred only in cold regions.

Owing to the lack of circulatory vessels and to conformation, the cornea and its region have a temperature sufficiently low to admit the growth of a mould, which would not be possible in ordinary tissues supplied by blood.

The disease—kerstomycosis—in the human subject is caused by moulds and is marked by corneal disintegration until it attains a crumbly surface, followed by necrosis of the cornea and hypopyon.

Aspergillus fumigatus is found to be the cause, though other moulds may also cause it.[7]

*(To be continued.)*

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XUM

# THE STUDY OF COCCIDIOSIS (EIMERIA CAVIÆ [Sheather 1924]) IN THE GUINEA PIG

## (Continued)

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### Degenerate Oocysts

Henry (1932) states that a striking feature of coccidiosis of the guinea pig is that, if no re-infestation occurs, the infestation experimentally given cures itself when the sexual cycle is completed, provided that it is not fatal. The diarrhœa ceases and the animal begins to gain in weight.

Further, she states that, when animals recovered in this way, whether the infestation was natural or experimental, they often passed degenerate oocysts in their fæces, the number of which increased until almost all the oocysts being passed were degenerate. They were passed in decreasing numbers for several weeks. These oocysts contained an amorphous, non-granular protoplasm, with large globules in it, or the globules only were present without any cytoplasm, or the cysts were smaller than is normal and were misshapen. She thought that these degenerate cysts resulted from the influence of some response made by an "immune," or partially "immunised," host on the gametocytes before the oocysts were formed, rather than on the fully formed oocyst.

Whether this interpretation is true or not, further work, done on clean animals reared artificially away from their mothers, must decide. It is worth considering, however, whether these degenerate cysts are not merely cysts which have been re-ingested and partly digested by the animal. If it should be shown that guinea pigs, like rabbits, eat their own fæces, such an interpretation would be more likely.

### Viability of the Oocysts

Earlier authors do not say anything about the length of time the oocysts of *Eimeria caviæ* can be kept in water or in any other fluid in which they may have been left to sporulate, before they cease to be capable of causing an in-

festation. The writer made no special experiments to test this point, but it is interesting to record that no difficulty at all was experienced in obtaining heavy infestations in guinea pigs by feeding to them oocysts when they were freshly sporulated, that is to say, 9-11 days after they had been collected from the fæces. The discussion below of the duration of the various phases of the life history shows that it is hardly possible, so long as we are ignorant of the nature of resistance to infestations with coccidia, to make precise statements about the variations in the viability of the oocysts. It may happen, for example, that oocysts which produce heavy infestations in some hosts will not, even so soon as seven days later, cause infestations in others and we have as yet no means of determining whether this is due to failure of the oocysts because they have lost their viability or to greater resistance of the guinea pigs. Guinea pig 136, for example, was not infested when it was given 1,259,000 oocysts, of which 566,550 had sporulated, although seven days earlier similar doses of the same batch of oocysts had produced heavy infestations in other guinea pigs. These oocysts had been kept during the seven days interval in the 1 per cent. formalin in which they had sporulated at laboratory temperature. On the other hand, it can be definitely stated that oocysts which had produced good infestations when they were freshly sporulated, no longer did so when they had been kept for 36 days in water after they had sporulated. The same was true when they were kept for 46, 50, 59, 61, 76 and 80 days in 1 per cent. formalin or water or in a 1 in 20 dilution of Milton with water.

In general, the writer's records show that good infestations will be obtained if oocysts are kept for two weeks or so in water, 1 per cent. formalin, or a 1 in 20 dilution of Milton, by which time 60-80 per cent. of them will normally have sporulated at laboratory temperature. They may even give good infestations if they are so kept for as long as three weeks. But their viability evidently decreases the longer they are kept, until four weeks or so after their removal from the fæces they are not likely to infest new hosts. It is understood, of course, that throughout the oocysts are kept moist. Drying, and especially the effect of sunlight on drying or dried oocysts, will, it is well known, rapidly kill them.

### Variations in Size of the Oocyst and Other Phases

In some of the colons examined a number of what appeared to be small merozoites were found, which gave rise to the impression that a second smaller species, or at any rate a smaller race of a constant size, might be present. The presence of a proportion of small oocysts in the fæces of most of the guinea pigs examined strengthened this impression.

Henry (1932) notes a smaller form of the coccidia she studied in a few of the guinea pigs she used. She does not record the size of these, but says that the oocysts of *E. caviæ* vary from 12.8-25$\mu$ in length by 12.8-22.4$\mu$ in

breadth. The smallest oocysts found by the writer measured 17.6 x 12.1μ, which is about the same size as the smallest that Bugge and Heinke record, namely, 17.4 x 12.2μ. Sheather's smallest were 17 x 13μ. Henry is therefore alone in finding oocysts smaller than 17 x 12.2μ. It is evident, however, that oocysts markedly smaller than the average size commonly exist and that the oocysts passed by all guinea pigs show, like the oocysts of other species of coccidia, a wide variation in size. The writer measured considerable numbers of them from freshly passed fæces and found that they varied from 24.2-17.6μ long by 19.8-12.1μ broad. Almost every possible transitional size between these extremes was found. Of 100 freshly passed oocysts counted, the average size was 19.33 x 16.5μ. The largest among this hundred was 24.2 x 19.8μ, the smallest 17.6 x 12.1μ; 60 per cent. of them measured 22 x 17.6μ, being oval; 2 per cent. of them measured 17.6 x 17.6μ, and 1 per cent. of them measured 18.7 x 18.7μ.

Most were thus oval, as Henry also found, some being spherical or almost so. Oocysts measured in sections showed a similar size variation, with similar intermediate grades of all varieties between the extremes, but the distortion due to fixation and staining of the sections made it more difficult to measure them accurately and shrinkage reduced their maximum size. Twenty oocysts fixed by Carleton's sublimate-formol mixture varied between 22-15.4μ long x 13.2μ broad; twenty fixed by Helly's mixture varied between 19.8-17.6μ x 15.4-13.2μ. Measurements of the sporoblasts inside the sporulated oocysts showed that these were much the same size in both the larger and smaller oocysts.

When the other stages of the parasite in the sections were measured it was found that a similar size variation was shown by them all and that they varied slightly in size when different fixatives were used. When formol-saline or Flemming's or Bouin's mixtures were used, the shrinkage of the tissues around the merozoites was so marked that the merozoites looked so strikingly small that they seemed to belong definitely to a smaller species. Measurements of them showed, however, that they were not actually much smaller than the merozoites in sections fixed by Helly's fixative, which often does not create a shrinkage space round them at all. Nor was their size significantly different from that of living merozoites scraped from the colon mucosa and fixed in smears in Schaudinn's sublimate-alcohol. The smallest merozoite bundles found in sections measured 6μ long and these, occurring in the largest shrinkage space seen, 16μ wide, looked smaller than others of the same size which either lay in smaller shrinkage spaces or had no shrinkage space at all around them. The merozoites measured in sections fixed by all fixatives varied between 6-16μ x 1.0μ thick, but only a very few were as small as 6.0μ long, and it is possible that these were not cut in the section through their whole length. The majority measured 8.0 to 14μ long, the average of a large number measured being in all fixatives 10.2μ. The length of living

merozoites fixed on smears by Schaudinn's sublimate-alcohol varied from 8.0 to 15$\mu$ with an average of 11.8$\mu$. These sizes agree well with the sizes given by Sheather, 10-15$\mu$ long x 1.5-2$\mu$ thick, and with Henry's " merozoite cysts " which were 10-16$\mu$ long.

A similar variation in the size of the full-grown female and male gametocytes was also found. Measurements of full-grown male gametocytes in sections fixed by all the different fixatives used showed an average size of 14.06$\mu$ long by 11.52$\mu$ broad, the length varying from 12.6-15.4$\mu$ and the breadth from 8.6 to 13.6$\mu$. Sheather (1924) states that the male gametocytes are very variable in size, some reaching " a maximum diameter," by which he presumably means a long diameter, of 25$\mu$.

The writer's measurements of female gametocytes showed an average size in all fixatives of 13.7$\mu$ long x 11.5$\mu$ broad, the length varying from 12-17$\mu$ and the breadth from 8.5-14$\mu$. Sheather (1924) gives the length of this phase as 16$\mu$. It must be borne in mind, however, that it is difficult always to be sure that only mature female gametocytes are being measured and that their breadth varies after their full length has been reached, because in the latter part of their development they do not grow longer, but only increase in breadth by the accumulation in them of the granules described below (Part II).

### Is There a Second Species?

All measurements of the developing phases of any organisms must be somewhat dependent on the observer's opinion whether the individuals measured are mature or not. They must, like the results of fæcal egg counts, be interpreted with discretion in the light of experience and by reference to other relevant observations. The measurements just recorded of the various phases of the life history of *E. cæviæ* do, nevertheless, show us that each of these phases shows considerable variation in size, even when allowance is made for the action of different fixatives. In this respect they are like the different phases of most other species of coccidia and all our experience of protozoology warns us that differences in size cannot be accepted as sufficient evidence of the presence of more than one species. Tyzzer (1929), in his careful study of the species of *Eimeria* infesting chickens, points this out and suggests that one reason for these variations in size is that large and small races within a species may be present and that these " size-races " may remain distinct even " when they develop together in successively infected intestines." Size-races of this kind are well known within the species *Entamœba histolytica* and in other species of protozoa.

Tyzzer (1929) suggests that the size of the organism may be influenced by " location in development," by which he must mean the actual site infested by the parasite and the host reactions it may encounter there, by the number of

XUM

organisms present, the stage of the infestation and by the age, breed and possible individual variations of the hosts. Other authors have emphasised these factors and, before we establish new species of coccidia, all the criteria set forth by Tyzzer (1928) should, as Andrews (1930) points out, be applied. The criteria set forth by Tyzzer are:

(1) the length of the period of development up to the discharge of the oocysts;

(2) The time required for sporulation at a given temperature;

(3) Host specificity or degree of restriction to a given host;

(4) Characteristic habitat or distribution of the organsim in the host;

(5) Cross immunity tests;

(6) Pathogenicity as determined by both experimental and natural infestation:

(7) Morphological studies, applied to the forms developing in the tissues as well as to the size and shape of the oocyst;

(8) The relation of the parasite to the host cell, together with the reaction of the latter.

When we apply these criteria to *E. caviæ* there is no reason at all to suppose that it represents more than one species of organism. Its variations in size can all be explained as the result either of differences in the actions of fixatives on the material examined or of variations in the vigour of different strains of the parasite, coupled with variations in the reactions of the host, whether these take the form of its normal physiological state or of "resistance" developed as the result of previous infestations, the existence of which has been proved by Henry (1932) and others. Larger and smaller races may, of course, exist, but these are a normal feature of the life history of many protozoa and have always to be taken into account.

There is, indeed, only one observation in the various studies that have been made of *E. caviæ* that could be legitimately adduced as evidence of the presence of a second smaller species of *E. caviæ*. Henry (1932) says that the smaller oocysts which she found sometimes predominated in particular guinea pigs and that, when this happened, the host showed "excessive œdema and gelatinous infiltration in the mesentery surrounding the first loop of the colon, with considerable quantities of mucus in the lumen of the intestine in this region, the mucoid masses consisting, for the most part, of packed small oocysts." She noted that, in three cases, the smaller oocysts appeared in the fæces about two days later than the larger ones. This gelatinous infiltration of the mesentery supporting the first loop of the colon was seen by the writer in a few guinea pigs, but it was never accompanied by a predominance of small oocysts. Sometimes the gelatinous infiltration was present in guinea pigs in

which no oocysts at all could be found. There was, therefore, no reason to suppose that this gelatinous infiltration was necessarily associated with the predominance of the small oocysts, so that even this feaure could not be adduced in favour of the view that a second smaller species of *E. caviæ* exists.

### Duration of the Various Phases of the Life History

It is difficult to make definite statements about the duration of either the whole life history or different parts of it, because these must depend on the size of the dose of oocysts which begin an infestation; on the effect on the oocysts of drying, sunlight, the length of time they have been out of the host in which they have been produced and other factors which may influence their viability; on the vigour of the strain of coccidia being studied; and on the resistance of the individual hosts. That individual hosts vary in their resistance to *E. caviæ* is likely, because Becker and Hall (1932) have shown that individual white rats vary in their resistance to *Eimeria miyairii* and all our knowledge of the resistance of animals to other parasites leads us to expect this. Variations in the size of the dose and the viability of the oocysts must be taken into account, because, when natural infestations occur, the host is usually infested by a slow and continuous intake of oocysts, which have been exposed to climatic and other factors which affect their viability, whereas, when experimental animals are artificially infested, they are usually given relatively large single doses of carefully preserved oocysts at certain intervals and the hosts are protected from infestation between the doses. We should expect, therefore, information based on these experiments to be useful only in a general way to the veterinary surgeon who has to control natural outbreaks of coccidiosis. It has already been explained, however, why it is very difficult to say that any guinea pig has never had an infestation with *E. caviæ* and therefore whether it is resistant or not. It seems likely that all guinea pigs must be regarded as resistant, and certainly the writer prefers to regard as resistant all the ones that he used.

The appearance of bundles of merozoites formed by the splitting of the schizont marks the end of the first schizogonous phase, and the earliest occurrence of this noted by the writer was in guinea pig 158, seven days after oocysts were fed to it, 81 per cent. of which, namely, 1,153,225, were fully sporulated when they were fed. The infestation produced was a very heavy one and the colon contained relatively few schizonts which had not yet split to form merozoites, most of them having split into bundles. There were also very numerous, very small trophozoites, which must have been, at a time so long after the oocysts were fed, derived from the merozoites already produced rather than from the sporozoites emerging from the oocysts. Guinea pig 158 was killed, in other words, just when the first generation of merozoites were

XUM

being formed and many of these had already infested new cells. Schizogony in this host required, therefore, 6-7 days.

Guinea pig 157 was given a smaller dose of oocysts from the same source (i.e., from the fæces of guinea pigs 157-162), 81.6 per cent. of which, namely 784,080, had fully sporulated when they were fed and the guinea pig was killed eight days after the dose. In spite of the much smaller dose (rather more than half the number given to guinea pig 158), this animal contracted an infestation practically as heavy as that of guinea pig 158, but it was at eight days rather less advanced, although it had lasted half a day longer. It consisted chiefly of bundles of merozoites and schizonts which had not yet split into merozoites, fewer very young trophozoites being present. Guinea pig 157 was thus probably the more resistant of the two. Guinea pigs 159 and 160, given approximately the same number of sporulated oocysts from a different source, showed much the same relationship, although 160 was killed at eight days and its infestation resembled that of guinea pig 157, while in guinea pig 159, killed 8½ days after its dose of oocysts, the infestation resembled that of guinea pig 158. In both of these guinea pigs, therefore, schizogony seemed to have required longer than it did in guinea pigs 157 and 158. It is unlikely that this happened because the strain of coccidia was feeble, because it produced a heavy infestation in both guinea pigs. It is more likely that they were more resistant than guinea pigs 157 and 158.

Oocysts from yet another source were fed to guinea pigs 125-130, but the number of oocysts fed was not counted. This was a vigorous strain, because in guinea pigs 128, 129 and 130 it produced heavy infestations. In 127, killed nine days after the dose, schizogony was still going on, but the sexual cycle had not yet begun. In 129 and 130 the same was true after 10 and 10½ days respectively. In 128, however, the sexual cycle was in full swing at 9½ days, plenty of male and female gametocytes being present, some of the male gametes being already mature. In 126, on the other hand, no infestation at all was produced and in 125 only a slight one was found which at eight days showed only asexual stages. It is more likely that the failure of the coccidia to infest these two latter ones was due to their greater resistance than to lighter doses of oocysts, but it is, of course, a defect of the observations just recorded that we cannot be sure of this. We know too little of the nature of resistance to be able to control it for experimental purposes.

This must apply, indeed, to all observations on the duration of the various phases of the life history. Reasons have already been given why it is difficult to say, even when apparently uninfested animals are used, that they have not had a previous infestation, from which a resistance has been developed. Nor have we any means as yet of controlling accurately either variations in the viability of the oocysts given experimentally or variations in the different strains of coccidia used. But for these very reasons observations like the ones just described probably indicate more closely what happens under natural

conditions than controlled experiments would, because natural infestations are produced by different strains of coccidia, whose oocysts vary in viability, in guinea pigs showing individual variations in their resistance to them.

We are therefore perhaps justified in concluding, even from the observations just described, that the first generation of merozoites is produced in some resistant hosts as late as 6-7 days after oocysts have been fed to them. In less resistant hosts, and in non-resistant hosts, if any of these exist, we should therefore expect them to be produced earlier. Because merozoites were still being produced in guinea pig 130 10½ days after oocysts had been fed to it and the sexual cycle had not then begun, we may conclude that schizogony can continue for some time longer than its normal duration, especially perhaps in guinea pigs that have been debilitated by an initial heavy infestation which has lowered their resistance. It is, of course, quite normal, as observation of almost any guinea pig will show, for schizogony to continue after the sexual cycle has begun, so that schizonts do not all suddenly begin to produce merozoites which become gametocytes.

In a similar way the sexual cycle begins at different times in different guinea pigs after the feeding of the oocysts. While in some of the writer's guinea pigs (e.g., 129) the male gamets were mature 9½ days after the oocysts had been fed, in others (e.g., 130) no gametocytes, either male or female, had been formed even after 10½ days. If, however, gametes are mature at 9½ days, we could expect that, in some guinea pigs, oocysts would be found in the tissues soon after this, at 10 days or so, and would appear in the fæces 10-11 days after the oocysts which began the infestation had been given to the host.

This period, namely, the time that elapses between the entrance of parasites into a host and their subsequent appearance in the blood or fæces has been called by Andrews (1926) the *prepatent period.* Sheather (1924), like the writer, found that it varied in the guinea pigs which he fed, but the shortest prepatent period observed by him was seven days and the longest 13 days. Henry (1932), commenting on this, says that " it is not in accordance with the more recent work on coccidial infections in general " to find so wide a variation in the prepatent period as 7-13 days, its duration being usually "exceedingly constant," varying not more than 12-24 hours. She discusses the factors which may influence its duration. Her own observations on 22 guinea pigs, none of which had any history of previous attacks of coccidiosis, showed that the first oocysts appeared in the fæces 11 to 12 days, but usually 11½ days, after oocysts had been fed to them. She does not say whether she determined the number of sporulated oocysts given or not. She suggests that oocysts found by Sheather before the eleventh day were derived from accidental infestations acquired within one to three days before he gave his infecting meal and remarks that another source of error may be the passage through the alimentary canal of accidentally ingested oocysts. She herself eliminated these sources of error, of which she had had experience during her earlier

XUM

work with coccidiosis of chickens, by carrying out only one experiment on the prepatent period at a time, i.e., by feeding oocysts only when no other guinea pigs which were passing oocysts were present. The writer's observations, admittedly few and not properly controlled, agree more nearly with Henry's than with Sheather's.

The evidence therefore is that, even in presumably resistant guinea pigs, the writer found that oocysts may appear *in the tissues* of the colon 10 days or so after sporulated oocysts have been fed by the mouth; that Sheather (1924) found them first appearing *in the fæces* as early as seven days or as late as 13 days after the infesting feed; and that Henry (1932) found that oocysts first appeared *in the fæces* 11 to 12, but usually 11½ days, after the oocysts were fed. Neither the writer, nor Sheather, nor presumably Henry, determined the dose of sporulated oocysts given when these records were made. The reasonable conclusion would seem to be that, having regard to the possible effects of the size of the dose, to possible variations in the virulence of strains of *Eimeria caviæ* and to the probable variations in the resistance of individual guinea pigs, the prepatent period may be as short as seven days and as long as 13 days. But Henry's (1932) view, based on her wide experience of coccidiosis in a variety of bird and mammalian hosts, that the prepatent period varies within 12-24 hours only, and her conclusion, based on experiments better controlled than those of either Sheather (1924) or the writer, that the prepatent period of *Eimeria caviæ* is 11-12 days, but usually 11½ days, must be given the consideration it deserves.

The *patent period* remains to be considered. This term was given by Andrews (1926) to the time that elapses between the appearance of parasites in the host and their disappearance from it. It therefore ends, in coccidial infestations, when oocysts are no longer found in the fæces of the host. It has already been pointed out that, in fæces of guinea pigs which have had an infestation with *Eimeria caviæ*, it may not be possible to detect, by direct examination or by a centrifugal flotation method, any oocysts for several days, but that a few oocysts may then appear for one day only and that similar successive negative periods of several days may follow, separated by isolated days on which a few oocysts may be passed. The writer also frequently found oocysts in sections of the colons of guinea pigs in the fæces of which oocysts could not be detected at all. It is evident, therefore, that it may be extremely difficult, if not impossible, to say that any guinea pig is not passing oocysts, so that it is very difficult, if not impossible, to say when coccidia disappear from the host, that is to say, when the patent period ends. It is doubtful, indeed, whether the use of the term "patent period," which may be useful in the study of some protozoal infestations, may not often be a handicap in the study of coccidiosis, because it may lead to the belief that, after 12 days or so, on the basis of Henry's observations, a particular guinea pig is free from coccidia, whereas the writer's records show that it may remain a carrier and

a source of the infestation of other guinea pigs for much longer periods than this.

**Situation of the Parasite in the Host**

All authors are agreed that *Eimeria caviæ* infests the colon rather than any other part of the alimentary canal. Labbé (1899) is content to say that they are found in the intestine. Strada and Traina (1900) are the only authors who have recorded coccidia from the liver of the guinea pig, and their paper does not make it clear that these were *E. caviæ*. They say that the coccidia they found were chiefly in the liver, where they occurred in small and often very numerous nodules, about the size of a pin's head or a millet seed, in the liver substance and on its convex and concave surfaces, but especially on the convex surface. They found coccidia in the intestine, but do not say which part of it was attacked. Bugge and Heinke (1921) found *E. caviæ* in the colon and do not record it from any other organ. They do not, as Sheather (1924) says that they do, record liver lesions. Probably Sheather was thinking of Strada and Traina's record of these. In only one of the guinea pigs which Sheather (1924) examined, he found a white spot on the mucosa of the gall bladder, but he could not find coccidia in this. He examined the entire intestines of eight guinea pigs, but did not find coccidia in the small intestines or livers of any of these. The usual site of infestation was the colon, especially the proximal end of it, the infestation decreasing as the anus was approached. When the infestation was severe, almost every cell of the mucosa of the colon contained a coccidium.

Henry (1932) also found that the colon is the chief site of the infestation. The cæca of two of her guinea pigs were, however, lightly attacked and the cæcum of one older guinea pig was infested more heavily, every cell being attacked over large areas of its mucosa. She found "a few isolated coccidia" in the small intestine of some guinea pigs, but no lesions occurred here and she never found coccidia in the stomach.

The writer found *Eimeria caviæ* in the colon only; it was never found in the small intestine, gall bladder or liver. Occasionally on the outside of the colon a few white spots were seen, but these were not characteristic and they were never seen on the liver. Serial sections made by the writer showed that the infestations were most severe at about 3-6 inches from the proximal end of the colon, although they were usually fairly severe also in the first three inches of it. When infestations were severe, the coccidia were numerous throughout the whole of the colon and occupied almost every epithelial cell in it, so that practically the whole epithelium was destroyed (Fig. 36). When infestations were slight, the coccidia were most numerous at the bottom of the crypts of Lieberkühn, but, when they were heavier, the parasites spread to the epithelial cells at the surface of the mucosa, where all stages of the life history could be found. *Eimeria caviæ* does not, like the species of

XUM

*Eimeria* of the chicken, described by Tyzzer (1929) and Tyzzer, Theiler and Jones (1932), show any marked preference for a particular site in the thickness of the mucosa. Very exceptionally oocysts have been found below the muscle layers, just under the peritoneum. (See Part II.)

**Pathology and Clinical Symptoms of the Disease Produced**

Strada and Traina (1900) record that the coccidia observed by them in guinea pigs could cause diarrhœa, with emaciation and death by tonic-clonic spasms. Bugge and Heinke (1921) and Sheather (1924) also described the effects that *Eimeria caviæ* can produce, but the best account of the clinical symptoms and pathological appearances caused by *Eimeria caviæ* is given by Henry (1932). She found that the first symptom shown by experimentally infested animals is diarrhœa. All her experimentally infested animals developed it, usually on the 11th-13th day after feeding oocysts, and it lasted four or five days, although in some animals it lasted only one day. When death occurred, diarrhœa was more severe and lasted until death. In some animals which revived, it lasted as long as it did in those that died. Constipation was also a constant symptom, being particularly noticeable about the eleventh or twelfth days, but rarely later.

Visible signs of illness usually began with the diarrhœa. Ruffled hair, contraction of the buttocks beneath the body, marked loss of appetite and polyuria were noted. Loss of weight sometimes accompanied the constipation and was marked when diarrhœa developed, amounting sometimes to a loss of almost half the body weight. With recovery and cessation of diarrhœa weight was regained, although such animals did not regain weight as quickly as normal animals. The asexual cycle of the parasite did not affect the temperature of the host, but this was sometimes subnormal just before death. Henry thought that the passage of visible amounts of mucus with the fæces indicated a bad prognosis and found large amounts of mucus, often packed with immature macrogametes, in the large intestine of animals that had died of coccidiosis. Only a few animals passed small amounts of blood with the fæces.

Internally, all the animals showing clinical symptoms of coccidiosis showed evidence of extreme emaciation. The lungs, liver, stomach and small intestine were normal. The large intestine was often distended with gas and was hyperæmic, its walls being rather thickened and the patches of lymphoid tissue in its walls especially prominent and often hæmorrhagic. Often small white and yellowish-white plaques were seen on the proximal half of the colon, especially in animals that had been ill for some time. Marked œdema was also noticed, and in a few animals the loop of mesentery supporting the proximal loop of the colon showed the gelatinous infiltration with marked œdema and enormous numbers of oocysts mentioned above. There were petechiæ in the mucosa and occasionally larger hæmorrhages. Sometimes the

whole inner surface of the proximal end of the colon was studded with pin-point hæmorrhages, and these extended throughout the colon and even to the rectum. The contents of the colon consisted chiefly of mucus, containing masses of oocysts, which were sometimes still surrounded by epithelial cells and sometimes immature forms of other stages were present still in the cells which they had invaded. Minute amounts of blood were only occasionally present, but the blood vessels were injected. The colon showed extensive infestation of the epithelial cells by the coccidia, extending into the crypts of Lieberkühn. When infestation was severe, practically the whole mucosa was destroyed, capillaries were sometimes ruptured, especially those just under the cuticle and "small cell infiltration" occurred wherever there were large numbers of coccidia.

Bugge and Heinke (1921) also noted the diarrhœa caused by the parasite. It was, they thought, more severe in young animals, in which it was sometimes hæmorrhagic, with considerable damage to the colon. In older guinea pigs they found no marked pathological changes, even when numerous coccidia were present. The writer can confirm this. There is often astonishingly little change in the tissues of the colon even when it is packed with parasites and one sees little else than injection of the capillaries. The appearance of encapsulation of masses of oocysts by connective tissue noticed by Henry (1932) was never seen. Theobald Smith (1910) noticed a similar encapsulation of oocysts in rabbits suffering from coccidiosis and suggested that it might be a means of disposing of the parasites and might be associated with "immunity." Possibly the infestations studied by the writer were never so severe as Henry's and he may have been observing less virulent strains of the parasite. Certainly most of the writer's guinea pigs showed practically no visible signs of illness, even when they were passing large numbers of oocysts.

### Pathogenicity and Mortality

Henry (1932) found that the symptoms described above always occurred in guinea pigs experimentally infested. Age did not influence the course of the infestation and males were as susceptible as females. She discusses the question of age in relation to "immunity." Nine out of twenty-two of her experimentally infested animals died, a percentage of 40.9, but she did not determine the number of sporulated oocysts given to them, so that it is difficult to assess the significance of this statement. She concluded that the mortality rate from *E. caviæ* is not so high as that caused by *E. tenella* of chickens, a conclusion with which everyone who has studied these two species will readily agree.

Sheather (1924) does not discuss the question of mortality, but Bugge and Heinke (1921) concluded that, although coccidiosis of the guinea pig may become severe and may even, especially in young animals, take on epidemic features, it is rarely by itself a cause of death.

XUM

This the writer also found. Guinea pigs that began to pass large numbers of oocysts had shown no visible signs of illness. At any time, however, whether an isolated guinea pig was passing small or large numbers of oocysts, or even none at all, an acute infestation might light up, especially in young animals. The guinea pigs usually recovered if they were well fed and well looked after. Death, if it occurred during such an infestation, probably resulted from some other cause, the coccidiosis being only a contributory cause which weakened the animal and rendered it less resistant to other diseases. *Eimeria caviæ* certainly does not cause tissue reactions in any way resembling those produced by the various species of *Eimeria* found in chickens and this is probably one reason why it is not in itself a cause of extensive mortality. Another reason is probably that it attacks the colon only, as a rule, and never attacks the duodenum, damage to which is much more serious for the host. Each species of the coccidia found in chickens produces, as Tyzzer (1929) and Tyzzer, Theiler and Jones (1932) have shown, tissue reactions which are so characteristic that they can be used as additional means of identifying them and many of them attack the duodenum and therefore are more pathogenic. Further, it is probable that *E. caviæ* has for a long time inhabited its host, so that host and parasite have become adapted to each other, whereas the species of *Eimeria* found in chickens have less recently established themselves in their host, which is therefore not so well adapted to them. Tyzzer's (1929) work shows clearly that the species of *Eimeria* found in chickens have not for long possessed the specific characters which he has recognised in them. The marked pathogenicity of some of them may be associated with the progress of this evolutionary process.

One of the primary causes of the death of some of the writer's guinea pigs, whether they were passing oocysts or not, was an acute duodenitis, with desquamation of the duodenal epithelium so severe that often large numbers of the villi were sloughed off entire. The duodenum was then full of desquamated villi, pus, epithelial cells and some blood, and the gall bladder was usually distended. The animal had a staring coat and evidence of fever. Bacteriological examination failed to find any cause of this condition, but coccidia were never found in the duodenum at any time. The condition may have been related to faulty feeding.

(*To be continued.*)

# SCRAPIE

## Observations on the Transmission of the Disease by Mediate Contact

By J. RUSSELL GREIG, Ph.D., M.R.C.V.S.

*Animal Diseases Research Association, Moredun Institute, Gilmerton, Edinburgh.*

THE disease of sheep known as scrapie represents a neurosis characterised by symptoms of intense and progressive pruritus, progressive asthenia and locomotor inco-ordination. The course, which usually extends over several months, almost invariably terminates fatally.

There has accumulated a large volume of evidence indicating that under natural conditions the disease can be transmitted from either the ram or the ewe to the immediate progeny; there is also evidence that such transmission results from congenital infection as distinct from hereditary diathesis and that the incubative stage of the infection is prolonged.

Several workers, including Dammann (1869), M'Fadyean (1918) and Bertrand, Carré and Lucam (1937), have attempted to transmit the disease artificially by the inoculation of sheep with material derived from typical cases of scrapie. All these experiments furnished negative results, but in recent years Cuillé and Chelle (1938) have produced evidence suggesting that scrapie can be transmitted by inoculation and that the ætiological factor is a filterable virus.

Dammann, M'Fadyean, Bertrand *et al.* also attempted to transmit the disease by placing healthy sheep in immediate contact with cases of scrapie. These experiments also proved negative; but it could be argued that they are inconclusive since the number of contact animals employed was small and since, especially in the experiment of Bertrand *et al.* and in that of Dammann, the periods over which the observations were made were relatively short.

During an extended study of the conditions of occurrence of scrapie in the field the writer had observed several instances which suggested the possibility that the disease could be transmitted through the medium of the pasture. It was found, however, to be a matter of extreme difficulty to obtain evidence in the field that the disease was thus transmitted since there so frequently existed the possibility that transmission had occurred by other means such as congenital infection or immediate contact. It was therefore resolved to attempt to determine, by means of the experimental method, whether scrapie could in fact be transmitted through pasture infection, and the following experiment was undertaken.

XUM

In November, 1932, the writer arranged at Moredun Institute a double-fenced enclosure, approximately one half acre in extent, in which all cases of scrapie admitted to the Institute were confined. On November 11 twenty Half-Bred gimmers and six pregnant Cheviot ewes were purchased from farms situated in districts of Scotland in which scrapie had not been known to occur. These sheep were placed in a field immediately adjoining the scrapie enclosure and thereafter approximately twice each week they were brought into the enclosure while the scrapie sheep were turned out into the field occupied by the contact animals, care being taken that during the process of interchange the two lots of sheep did not come into immediate contact with each other. The ewes were served in November, 1933, by a Cheviot ram lamb (born at Moredun on April 18, 1933), the progeny of one of the Cheviot ewes, and they were again served by this ram in November, 1934. There was, incidentally, a heavy death rate in the lambs of both these lambings.

Throughout the course of the experiment a few—usually three or four—cases of scrapie were maintained on the experimental pasture.

At the conclusion of the period of experiment and observation at Moredun (November 16, 1935) no case of scrapie had been observed among the contact sheep and these were then transferred for further observation to a farm in Midlothian (Farm M.) on which scrapie had not been known to occur.

The flock then comprised sixteen ewes (twelve Half-Breds and four Cheviots) and nine ewe hoggs—twenty-five in all; eight Half-Bred ewes and two Cheviot ewes having died during the course of the experiment from causes unconnected with scrapie while the wether lambs had been disposed of.

Soon after arrival at Farm M. three hoggs died in a condition of debility, the precise cause of which remained undetermined, but no case of scrapie was observed in any of the contact sheep until February, 1936, a period of approximately three years and three months after the commencement of the experiment.

Between February, 1936, and May, 1938, when the experiment was concluded, nine other contact ewes, representing both the Half-Bred and the Cheviot flocks, developed scrapie. In three of the ten cases (Nos. 5, 7 and 9) the diagnosis was not precisely determined, but the remaining seven were typical cases of the disease, and it was concluded that the investigation afforded experimental evidence indicating that scrapie is an infective disease, that the incubation period is prolonged, and that the causal agent can be transmitted through the medium of pasture.

*Case* 1.—In February, 1936,* scrapie was diagnosed in a pregnant Half-Bred ewe (one of the lot purchased in November, 1932) which had been served

* It will be noted that the precise date upon which the disease became manifest is not recorded in several of these cases. The flock was under the supervision of an observant shepherd, but as the onset of attack is insidious, an indefinite period—a few days to two or three weeks—may elapse before even a tentative diagnosis can be reached.

before leaving Moredun. The disease ran a typical course, and terminated in death on March 9. The medulla and spinal cord were examined for the presence of vacuolation in the nerve cells, and vacuoles typical of those now regularly found present in scrapie were observed.

*Case* 2.—On June 15, 1936, a Half-Bred ewe (one of the lot purchased in November, 1932) with twin lambs at foot developed scrapie. After a typical course death occurred on August 17. Vacuolation present.

*Case* 3.—On August 26, 1936, a gimmer (born at Moredun in May, 1935), the progeny of the Cheviot ram and one of the Half-Bred ewes developed scrapie. She had lambed the previous May, but the lamb had died shortly after birth. After a typical course death occurred on September 15. Vacuolation present.

*Case* 4.—About the beginning of September, 1936, a Half-Bred ewe (one of the lot purchased in November, 1932) was observed showing signs of skin irritation. The ewe was transferred to Moredun on February 3, 1937. The symptoms of scrapie were gradual in their development, and the course extended throughout approximately fifteen months when it terminated in death on November 22, 1937. Vacuolation present.

*Case* 5.—On March 22, 1937, it was reported that a Half-Bred pregnant ewe (one of the lot purchased in November, 1932) was acutely ill. The ewe was transferred to Moredun where the case was diagnosed as pregnancy toxæmia. A few hours after admission to the Institute the ewe was destroyed *in extremis*. It was then reported that symptoms suggestive of scrapie had been in evidence in the ewe at Farm M. "for some time." Vacuolation present.

*Case* 6.—In March, 1937, a Half-Bred ewe (one of the lot purchased in November, 1932) developed pregnancy toxæmia, but during the attack gave birth to triplet lambs whereupon she recovered completely. Shortly afterwards she developed symptoms of scrapie. The ewe was transferred to Moredun on March 26 and was destroyed on May 11. In this case no vacuolation of nerve cells was detected.

*Case* 7.—On March 24, 1937, a pregnant Half-Bred ewe (one of the lot purchased in November, 1932) was transferred to Moredun from Farm M. as having developed scrapie. She was kept under observation and although symptoms suggestive of scrapie were evident no precise clinical diagnosis was reached. On May 3 she developed typical symptoms of pregnancy toxæmia and was destroyed *in extremis* the following day. Vacuolation present.

*Case* 8.—In the beginning of July, 1937, a Half-Bred gimmer (born at Moredun in April, 1935) with lamb at foot developed symptoms of scrapie. The ewe was transferred to Moredun on July 16, 1937. The disease ran a normal course and the animal was destroyed on November 26. Vacuolation present.

XUM

*Case* 9.—In November, 1937, it was reported that a Half-Bred ewe (one of the lot purchased in November, 1932) had developed symptoms of scrapie at Farm M. The sheep was transferred to Moredun on December 4, 1937, but died two days later without a definite diagnosis of scrapie having been reached.

*Case* 10.—In January, 1938, a Cheviot ewe (one of the lot purchased in November, 1932) developed the initial symptoms of the disease. On February 9 the ewe was transferred to Moredun where the disease became manifest in its typical form. The ewe was destroyed *in extremis* on May 17, 1938. Vacuolation present.

The experiment was thereafter terminated.

Of these ten cases, seven may be accepted as those of scrapie; the remaining three (Nos. 5, 7 and 9) should, it is considered, be regarded as probable but doubtful cases of the disease.

## Discussion

It was at one time believed by flockmasters that mediate contagion through the pasture did not occur since in practice it was found that the disease could be eliminated from a farm by selling off the whole flock and shortly thereafter replacing it with healthy stock. The period during which pastures remain infective is, however, unknown; it may be of short duration. It is also true that in a considerable number of instances the procedure of complete restocking has failed to eradicate the disease in so far that, although it has resulted in a marked reduction in the incidence of scrapie, the disease has in due course reappeared and progressively increased in incidence. This is especially so in the case of lowland farms on which the pasture is relatively intensively grazed as distinct from sparsely stocked hill farms on which the chance of contracting infection from the pasture would be much reduced.

## Summary

There is adduced experimental evidence indicating that scrapie is an infective disease, that the incubative period is prolonged and that the causal agent can be transmitted mediately through the pasture.

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# SOME REMARKS ON TWO DISEASES AFFECTING THE LUNGS AND AIR-SACS IN POULTRY

By C V. WATKINS, M.R.C.V.S., D.V.S.M.

1. Air-sac Mites (*Cytoleichus nudus*).

THESE parasites of poultry have usually been deemed of little economic importance. Most textbooks on poultry diseases refer to them as parasites, but otherwise describe them as harmless. SEYFARTH, *Deuts. tierarztl. Wschr., 47,* 289-291, demonstrated in a R.I.R. hen lung lesions, numerous mites in smears from the trachea, lungs and air-sacs, together with intestinal catarrh and inflammation of the oviduct. Because of the occurrence of the lung lesions he considers that *C. nudus* cannot be considered as harmless even in moderate numbers.

Such a finding substantiates some of my own post-mortem findings. From September, 1933, to September, 1937, heavy infestation of the lungs and air-sacs was found in nine fowls. *All the cases were associated with avian tuberculosis.* Usually the losses occurred in poultry kept on general farms under rather poor conditions of husbandry. In many cases the birds used old cart sheds, etc., in which to roost. During the year ended September, 1937, one case of air-sac mite disease was observed without concurrent tuberculosis, and this was my first record of an uncomplicated case in my area. Between Septem, 1937, and September, 1939, twelve cases of infestation by *C. nudus* were determined and only half of them were complicated by tuberculosis. During the current year another uncomplicated case has been found.

The important features of this series of findings are:

1. The common association of tuberculosis with air-sac mites. In this connection it is interesting to note that tuberculosis of the lungs often occurs in these complicated cases, both the chitinous membranes of the mites and *M. tuberculosis* staining well by the Ziehl-Neelsen technique for sections.

2. Uncomplicated cases of air-sac mite disease have been determined with much greater frequency during the last three years.

3. In general, the conditions of poultry keeping of affected flocks have not been good.

Several points about the disease still await solution. The exact mode of infestation of the common fowl is not known. It appears that association with wild birds may have some bearing on this problem. It has been impossible to determine whether air-sac mites or tuberculosis caused the primary

disease in the complicated cases. There is also the possibility that the mites may (a) serve as vectors in the spread of tuberculosis in affected flocks, or (b) render poultry more liable to contract tuberculosis through their irritant and, according to Seyfarth, pathogenic action upon the lungs. One or other of these assumptions appears to be justified, seeing that the lungs of poultry apparently escape infection in most uncomplicated cases of avian tuberculosis, probably because the usual route of infection is by ingestion.

2. Brooder Pneumonia or Aspergillosis of Chicks.

During the two-year period ended September, 1939, it was a routine practice in my laboratory to make a cultural test for pathogenic moulds on malt agar slopes from the lungs of chicks of upwards of 14 days old and in which chill was suspected to have caused losses. At the same time a cultural test for bacillary diseases was carried out and all suspicious lesions were noted. The number of cases of brooder pneumonia in this age group of chicks was as follows:

| | Number of groups of chicks tested. | Cases of Aspergillosis determined. |
|---|---|---|
| From Oct. 1, 1937, to Sept. 30, 1938 ... ... | 144 | 14 |
| From Oct. 1, 1938, to Sept. 30, 1939 ... ... | 150 | 16 |
| Totals ... ... | 294 | 30 |

It will be seen that the incidence of this form of disease in chicks is approximately 10 per cent. of cases examined and that very little variation has occurred from one year to the other. The figures may not be a correct estimate of incidence of brooder pneumonia throughout the country. They apply to chick rearing in Devon and Cornwall and only to chicks up to 14 days old. They are sufficiently high, however, to justify a continuance of such a routine examination because, in my opinion, control measures have to be more thorough than against bacterial diseases such as "B.W.D." To mention one item of variable importance, food and litter are rarely taken into consideration in the control of "B.W.D."

Depending on the circumstances, it has been found possible to determine the source of the outbreak in a proportion of cases. The actual food used, the food storage accommodation (food bins, etc.) or the litter may each or all be at fault. Disinfection must take these into account together with the diseased group of chicks, the chick quarters and all apparatus in use. Attention paid to the soiled floor of the house with its litter is not sufficient and the whole of the interior of the house, especially ledges, should be disinfected.

I have gradually become more convinced that this disease is of major importance. I carry out post-mortem examinations on about 1,200 head of poultry each year together with groups of chicks. Until about two and a half years ago, when a routine cultural test for aspergillosis was commenced, I feel certain that many outbreaks remained undiagnosed in chicks and were reported as "showing evidence of chill" in the majority of cases. Such a term probably covers numerous chick diseases relative to their mortality, but occasional reference to the old agar slopes used for cultural test of "chilled" chicks revealed a growth of *Aspergillus spp.* on some of them. Further evidence of this disease was also suggested by the high mortality that occurred in some outbreaks. Very often only a few chicks remained alive out of groups of upwards of 100 chicks. In extreme cases of which I have several records losses were 100 per cent. One of these latter outbreaks occurred in 1933 in pedigree turkey poults of several breeds, many of the stock birds being of prize-winning and championship class. Altogether about 200 poults, comprising all attempts at rearing on these premises, were lost before the owner got in touch with me. Diagnosis then lead to incrimination of badly stored litter.

Aspergillosis may occur as a chronic air-sac disease in adult birds. I have noted air-sacs thickened by a cheesy deposit and inflammatory reaction to a depth of one inch in a bronze turkey cock. The interior of the affected sacs was a mass of green sporulating mould.

Another reason for treating the disease as of major importance is that it is a potential source of danger to the attendants.

In my records of these cases several interesting findings have emerged. The most important is that only a proportion of affected chicks showed "typical" macroscopic lesions of aspergillosis. The "diagnostic" findings in small chicks are cream-coloured nodular lesions in the lungs combined with similar lesions of the air-sacs and inside the keel bone. The keel bone lesions often have a flattened appearance. All nodular lesions may be extremely small or, in older chicks, may attain the size of a pea. The lungs often appear congested in cases in which the above lesions are absent. When lesions are present, confirmation of the disease may be made by (a) stained smear preparations from the lesions, (b) cultural tests on malt agar slopes, or (c) stained sections of tissue. Toluidine blue or thionine blue stains serve equally well for this purpose and for the direct smear preparations. Of these the cultural test is the only certain method.

Another important finding was that many chick losses actually occur as a result of very complicated and intercurrent disease processes. The following table may be of interest as it shows the relationship between a tentative diagnosis according to post-mortem lesions and the actual cultural and parasitological findings. The table is given with the express purpose of

XUM

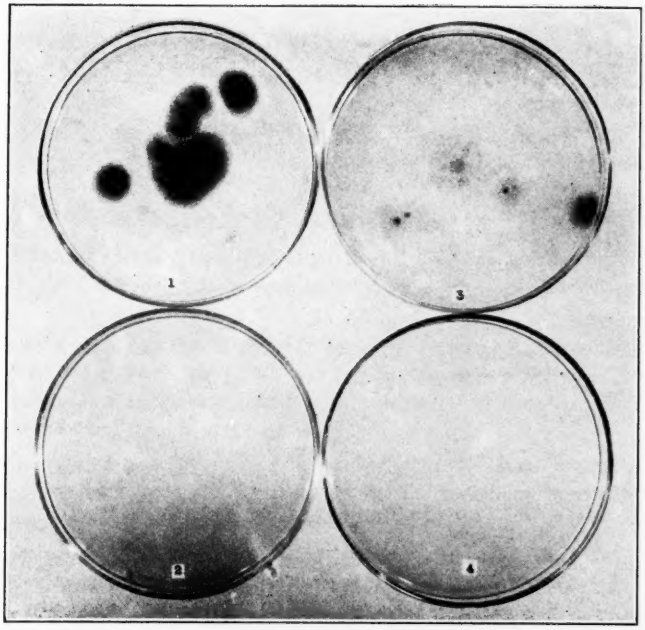
emphasising the fallacies of "diagnosis" based on macroscopic lesions in poultry generally.

| Tentative Diagnosis. | Aspergillosis. | "B.W.D." | Coliform Infection. | Cæcal Coccidiosis. | Aertrycke Infection. |
|---|---|---|---|---|---|
| 1937 to 1938 | | | | | |
| Aspergillosis, 4 cases ... | 4+ | 1+ | | | |
| Slightly suspicious of Aspergillosis, 1 case ... | 1+ | | | | |
| "B.W.D.," 5 cases ... | 5+ | 5+ | | | |
| "Chill," 3 cases ... ... | 3+ | | 2+ | | |
| Acute Cæcal Coccidiosis, 1 case ... ... ... | 1+ | | 1+ | 1+ | |
| 1938 to 1939 | | | | | |
| Aspergillosis, 4 cases ... | 4+ | | | | |
| "Chill," suspicious of Aspergillosis, 1 case ... | 1+ | | | | |
| Aspergillosis and "B.W.D.," 2 cases ... ... | 2+ | 2+ | | | |
| Aspergillosis and Acute Cæcal Coccidiosis, 1 case ... ... ... | 1+ | | | 1+ | |
| "B.W.D.," 4 cases* ... | 4+ | 4+ | | | |
| "Chill," 2 cases ... ... | 2+ | | | | 1+ |
| Acute Cæcal Coccidiosis and "Chill," 2 cases ... | 2+ | | | 2+ | |

* One of the above cases which appeared to be quite typical of "B.W.D." by the macroscopic lung lesions was especially interesting. "B.W.D." was proved by cultural tests, etc. Sections of the lung were cut for demonstration purposes. Only when examined microscopically were lesions of aspergillosis noted side by side with those of "B.W.D." Owing to an oversight no cultural test for aspergillosis had been made in this case.

Another important feature of these 30 recorded cases is that more than half the outbreaks were complicated by bacterial disease of which "B.W.D." was implicated in 12 cases. Such an association may account for some of the very variable mortality rates of "B.W.D." and for a continuance of losses in the absence of any specific and concurrent examination for aspergillosis.

The optimum temperature for growth of the pathogenic species of *Aspergillus* so far isolated appears to be 37.5 deg. C. Good growth occurs within 48 hours on malt agar slopes or plates, but diagnosis is usually possible after 24 hours' incubation. The illustration shows 48-hour growth on malt agar plates of two strains of *Aspergillus spp.* (probably *A. fumigatus*) at 37.5 deg. C. (Nos. 1 and 3) and at room temperature (Nos. 2 and 4).




Cultures of two strains of *Aspergillus spp.* (probably A. fumigatus) on malt agar plates.*

Nos. 1 and 3 were incubated at 37.5° C. for 48 hours.
Nos. 2 and 4 were incubated at room temperature for 48 hours.

* Malt agar prepared as follows:—
Allen & Hanbury's Malt Extract 3 to 3.5% and Agar 2% in water.
Dissolve agar in half the water in steamer.
Dissolve malt extract in other half of hot water.
*pH* should be between 5.0—6.0.
Steam for ½ to 1 hour. Fill into tubes and autoclave for 20 min. at 1 atmosphere pressure.
If *pH* requires adjustment use Lactic or Tartaric Acid.

XUM

## Clinical Articles

# THE EFFECTS OF CHILL ON A MARE

By C. W. PEMBREY, M.R.C.V.S.
*Preston.*

*Subject.*—A Shire mare, about twelve years old.

*History.*—The mare was thought to be in foal, and was brought up from the fields about mid-December and tied up in a stable. She was not given exercise.

I was called on the night of January 7 to the mare, which was uncomfortable and sweating. She was striking up at her belly and crouching occasionally. Her temperature was 102.5 and pulse weak. She had dunged and obvious bowel sounds were heard.

*Diagnosis.*—The weather had been bitterly cold for over a week, and I attached much importance to the fact that the mare had not been exercised at all. I diagnosed, not colic, but the effects of chill.

*Course and outcome.*—The mare seemed to be easier the next morning, but was suffering from profuse diarrhœa. That night I was called to her again as she had become very restless and was sweating badly. The owner confidently predicted she would not live till morning. However, the following morning she appeared to be better, but she had eaten nothing nor drunk water. The diarrhœa had ceased, but wind was passed intermittently accompanied by a strong fœtid smell. The temperature was now 98 deg. On the fourth day the mare nibbled a little bran mash, and on the fifth, passed a little dung, but during the next few days she ate and drank very little. The temperature remained two to three degrees below normal. Symptoms of jaundice appeared during the third week and the mare dropped dead on February 1.

### Treatment

(1) *Medicinal.*—A respiratory and general stimulant was given on January 7, and a hypodermic injection of morphia gr. ii when the mare was so restless the following evening.

Subsequently, sodium bicarbonate, nux vomica, ammon. carb., a weak solution of hydrochloric acid and potassium nitrate in small doses were given in sequence.

(2) *General.*—More importance was attached to this than to medicinal treatment.

The mare was moved to a loose box and allowed to have her head the morning following my first visit. She was thoroughly wiped down twice daily and rugged up. She was drenched twice daily with two or three eggs beaten up with milk, or occasionally with gruel. During the second week she was walked out when there was sunshine and not too keen a wind, and twice was taken into a field where she showed sufficient interest to nibble some grass not covered with snow. However, this restricted exercise out of doors had to be discontinued during the third week owing to increasing weakness.

*Post-mortem.*—This revealed congestion and discoloration of the lung tissue, but not hepatisation, inflammation and degenerative changes in the pericardium, liver, kidneys, and bowels, there was the yellow tint of jaundice throughout the carcase. The mare proved to be not in foal.

*Remarks.*—The weather was bitterly cold almost throughout, and was not conducive to recovery. I heard that a mare on another farm had been lost under similar circumstances. I am convinced that had this mare been given daily exercise she would never have contracted the fatal chill. To stand a heavy mare in a stable without exercise in cold weather would appear to be more dangerous than leaving her to roam the fields.

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# HÆMORRHAGE AND PROLAPSE OF THE UTERUS

**By C. W. PEMBREY, M.R.C.V.S.**
*Preston.*

*Subject.*—A cross-bred Shorthorn heifer, about two years and three months old.

*History.*—I was called at about midnight to a "calf-bed" (prolapsed uterus). The farmer had had to pull the calf away and the uterus had followed almost at once. I found a small heifer in spare condition. She was soon hoisted across a bale of straw, her forelegs hobbled, and her hindquarters raised in the usual way.

After rinsing the uterus thoroughly with warm water and Weale antiseptic, I proceeded with its reduction, the uterus being slung and held up in a towel. The heifer being so young and small, the operation was not easy.

The uterus was about half-way back when, perhaps due to momentary

rough handling under the difficult circumstances, one of the "roses" (cotyledons) began to bleed in a steady stream. The owner at once suggested slaughter, but, taking into account the difficulty of getting a butcher at such an hour and the spare condition of the heifer, I persuaded him to take the chance of the heifer responding to treatment. This was no time for the niceties of surgery, and the owner was sent in for some string while the hæmorrhage was checked by pressure of finger and thumb. A piece of string was tied round sufficiently light to check bleeding. However, this became dislodged on the further manipulation to pass the affected cotyledon beyond the lips of the vulva, and bleeding recommenced. Another piece of string was then tied on with the same sequence of events, and a third and fourth piece tied in position until a tassel of strings hung from the cotyledon. However, the uterus was eventually replaced without further loss of blood, the membrane smoothed out, and the vulva pinned and laced. At 2.30 a.m. the heifer was sitting up quite comfortably, with her hindquarters raised, and no sign of straining. She was seen again two and three days later, the pins being removed on the third day. On visiting the farm about a month later the heifer was found to have made an excellent recovery without "whites" (leucorrhœa) supervening. I have not seen her since, the farm being a small one, ten miles away and not often visited.

*Remarks.*—The value of recounting this experience lies, to my mind, in showing from what a young heifer, and perhaps a cow, may recover after very severe hæmorrhage. It may encourage practitioners of little experience of such cases not to resort to the butcher without due regard to the possibility of successful treatment.

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# A PATHOLOGICAL CANINE FREAK

By C. E. STUART SCOTT
*Final Year Student, Bridgwater.*

*Subject.*—Spaniel bitch, 13 years old.

*History and Course.*—Two years ago the owner noticed a small swelling just in front of the two forelegs. This swelling remained quiescent for 18 months, and for the following six months it gradually increased in size and extended perpendicularly almost to the ground as a pedunculated growth. At this stage the dog was brought to the surgery and the tumour was excised.

On cutting into the growth about two ounces of serous fluid escaped. The

wall varied from an ⅛ in. to ¼ in. in thickness and appeared to be purely fibrous in nature.

*Remarks.*—The interest of this case is the length of the growth from its base and, although it dangled like a pendulum between the forelegs, the animal had become so accustomed to it that it did not interfere with locomotion.

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# A CASE OF VITAMIN C DEFICIENCY

By MARJORIE G. JORDAN, M.R.C.V.S.
*Dorking.*

THE subject was a five months Cairn bitch puppy. Her previous history was normal, except that at the age of three months she was reported to have cried out nervously two or three times when the mouth was touched or approached. She was normal in every other way, and the trouble was thought to be due to teething.

At five months old she was brought into the surgery with a very swollen and painful lower jaw, the left ramus being particularly thickened, and there appeared to be a hard, bony enlargement at the site of the thickening. There was a discharge of pus and blood from the gum below the left molars. The mouth was partially open, and there was considerable salivation and a very foul odour. There was no temperature, and the appetite remained good.

A foreign body or deep-seated abscess was suspected, and an incision was made over the enlarged area. There was some hæmorrhage, but nothing was found. The wound was well swabbed out, and the condition entirely cleared up during the next few days, and the puppy was sent home.

Three days later she was sent in, as the jaw had enlarged again, and at the same time a large abscess was found above the left shoulder. This was lanced and dressed and healed up rapidly, and the jaw swelling subsided simultaneously. It was decided to ask Mr. Ede to X-ray the jaw. The result showed what appeared to be a normal jaw. Dr. McCunn was consulted and gave as his opinion that there was rarefaction at the site of the lesion, and it was probably a case of rarefaction of bone due to a deficiency of vitamin C—a condition corresponding to infantile scurvy in the human subject.

The puppy was put on to concentrated vitamin C in the form of "Ceetamin" tablets, fresh fruit juice and Ostelin. The jaw gradually became quite normal and there was no recurrence of the condition.

The owners bought the pup at eight weeks, and from that time her diet had consisted of raw beef, brown bread, cod liver oil, and lactol.

It is usual in these cases to get acute pain in the legs and lumbar region

as well as in the jaw. Here, I might quote extracts from the record of a case sent me by Major L. P. Pugh, of Sevenoaks :—

"A West Highland bitch pup aged three months was brought in with intense pain localised to the femur of the right hind leg. X-ray for suspected fracture was negative. As local treatment with heat produced no alleviation of the pain and screaming, I anæsthetised and cut down on to what I thought might be a deep-seated abscess.

"Eventually I discovered a large sub-periosteal hæmatoma. I incised this, and completed the operation by closing the wound. The next morning the puppy was sound and cheerful. Five days later, the puppy again began to scream when approached—and the left ramus of the jaw was found to be thickening and very painful. After two days, both rami of the jaw were swollen and very painful, and Barlow's Disease or infantile scurvy was suspected.

"Large doses of fresh lemon juice were given, and there was an almost immediate alleviation of the symptoms, and at the end of a fortnight the jaw was normal.

"The pup's diet had been well cooked beef and puppy biscuits, plus a plentiful supply of *powdered* milk."

## Abstract

# BARLOW'S DISEASE OR VITAMIN C DEFICIENCY IN THE DOG *

SINCE the identification of vitamin C in 1932, much research has been inaugurated to determine the effects of a deficiency of this vitamin in various animals and to evaluate methods of combating such deficiency diseases.

At the present time it appears that in the animal world, the dog, the pig, the monkey, and the guinea-pig are susceptible to a deficiency of ascorbic acid, while the ruminants, the solipeds, the birds, and the rat are capable of synthesising this vitamin themselves and are not affected by a vitamin C-deficient ration.

A deficiency of vitamin C in the young dog causes a condition known as Barlow's disease.[1] The symptoms are clear. Not only signs of rachitis frequently are present, but the young dog, although in good condition, remains

* Reprinted from the *North American Veterinarian* (1940), *21*, 164.

nonchalant and is most frequently in a recumbent position. It cries when it is touched. The pain is especially evident during pressure on certain bones, such as the radius in its inferior third and the tibia in its superior third : in the points of junction between the diaphysis and the epiphysis, subperiosteal hæmorrhages are the cause of the violent pains during the movements which pull about the altered periosteum. The animal is anæmic.

An examination of the urine shows a very slight renal excretion of ascorbic acid (vitamin C)—perhaps 2 to 5 milligrams per litre—as compared to a renal excretion of 25 to 30 milligrams per litre in the normal dog. This lowered excretion clearly indicates a vitamin C deficiency in the animal.

Treatment consists of the administration of massive doses of the deficient element. Lemon juice or commercial ascorbic acid is used. Dosage (case reports concern animals six months of age and younger) ranges from 50 to 100 milligrams of ascorbic acid and/or the juice of one or two lemons. Vitamins A and D may be given. The symptoms disappear slowly.

In some animals the disease is benign and ascorbic acid quickly relieves the trouble. However, in other animals the disease does not result exclusively from a deficiency of this vitamin in the ration but rather from a profound disturbance in the cycle of ascorbic acid metabolism in the animal, since the administration of massive doses of vitamin C does not effect recovery of a rate of renal excretion found in normal animals. In these patients the deficiency is serious and remains in spite of administration of vitamin C.

REFERENCE

(1) Gregoire, C.: La vitamine C et la maladie de Barlow chez le chien. (Vitamin C and Barlow's disease in the dog.) *Ann. Med. Vét., 83* (1938), 366-371.

# Abstracts of Current Literature

**The Rapid Detection of Mastitic Milk by a Portable Electrometric Device.** By E. C. McCulloch (1940). *Vet. Med.*, XXXV, 158.

THE author describes an electrical apparatus and its use in detecting cases of mastitis. His work is based on the assumption that the electrical conductivity of abnormal milk is greater than that of normal milk. It is stated that "a factor which probably has deterred the development of simplified methods for detecting mastitis by electrical conductivity is the rapid alteration of the physical composition of milk which results from the passage of continuous electrical current through it." The apparatus devised by the author can be "carried readily from cow to cow, with the readings made in a minimum time and the results immediately known to the examining veterinarian."

The apparatus is described in detail and is illustrated. The principle of the apparatus is that of the Wheatstone bridge—the milk cell replacing one arm of the bridge. Earphones form part of the equipment. It has been found that the electrical conductivity is a good indicator of milk from abnormal quarters. Early cases of mastitis can be detected with a degree of accuracy comparable to that found by the methods usually adopted in the laboratory. Over 1,000 quarter samples have been tested by both methods.

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**Veritol: a Blood-pressure Stimulant for use during and after Operation.** By H. Dodd and G. Merton (1939). *Brit. Jl. Surg.*, XXVII, 78.

VERITOL—a drug, chemically related to hordenine and tyramine—has been found reliable and satisfactory for restoring the blood pressure during and after operations in human beings. No injurious effects were produced and the rise in the pulse rate was never excessive. A dose of 1 c.c. given intramuscularly was usually used, though 1.5 c.c. was not found excessive. The action starts in one to five minutes, usually three minutes, after injection, reaches the maximum in about 18 minutes, and then falls gradually over the next 30 minutes.

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**Narcosis in the Pig and Dog following the Intravenous Injection of "Eunarcon."** By V. Calisti (1939). *Boll. d. Soc. Eust.*, XVIII, 173. (Summary translated.)

THE author has carried out experiments on the action of Eunarcon on pigs and dogs by intravenous injection as a narcotic in surgical operations. He obtained good results with the following doses:

Pigs from two to four months old—0.15 c.c. per kilo.

Older pigs—0.2 c.c. per kilo.

Dogs—0.3 to 0.4 c.c. per kilo.

Some excitement was observed while the dogs were becoming conscious. This was prevented by the subcutaneous injection of morphine ½ to ¾ hour before the drug was administered.

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**The Feeding of Seaweed Meal to Horses.** By A. Moholdt (1940). *Norsk Veter.-Tidsskt.*, LII, 38. (Author's Summary in English.)

AN account is given of experiments in feeding with seaweed meal ("Algit" tang meal) carried out on Eastland horses during military service.

Altogether 33 horses took part in the experiments, ten of them as controls. The experiments lasted two months.

The results showed that a proportion of the oats (20 per cent.) can be

replaced by seaweed meal without causing any demonstrable injury or impairment of the animals' condition.

The excrement was examined for presence of parasite eggs. It was found that the horses fed on seaweed meal had far fewer eggs of parasites (Sclerostomum, Ascaris) than the control animals.

The feeding experiments were of orientative nature. They will be continued.

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## ERRATUM

In Volume 96, No. 2 of THE VETERINARY JOURNAL, on page 72, appeared an abstract of an article, "A Survey of Tubercle Bacilli Types and of Type Diagnosis," by H. Holth (1939), *Skand. Veterinar.-Tidskrift.*, XXIX, 1125.

In the abstract it is stated that "The avian type produces no results in rabbits or guinea-pigs, but in fowls the progressive disease develops." This should read "The avian type produces no results in guinea-pigs, but in rabbits and fowls the progressive disease develops."

The use of the rabbit in typing the avian strain of tubercle bacilli is, of course, well known.

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